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Original Article

Sleep disturbance and cardiometabolic risk factors in early pregnancy: a preliminary study ☆

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ABSTRACT

Background: Cardiometabolic (CM) risk factors are linked to increased morbidity. Disturbed sleep is associated with CM risk factors in late pregnancy, but little is known about sleep in early pregnancy and CM risk factors.
Methods: Diary and actigraphy-assessed sleep information, as well as CM outcomes (blood pressure (BP) and body mass index (BMI)), were collected thrice from pregnant women ($N = 161$) in early pregnancy: T1 (10–12 weeks), T2 (14–16 weeks) and T3 (18–20 weeks). The sleep variables evaluated included sleep onset latency (SOL), wake after sleep onset (WASO) and total sleep time (TST). Sleep variables were dichotomised using established clinical cut-offs.
Results: BMI and BP significantly changed across time. Women with persistent SOL $\geqslant$ 20 min had greater BMI than women without persistent SOL $\geqslant$ 20 min prior to covariate adjustment at T1 and T2, but at T3 the BMI values converged. Similar results were observed for persistent WASO $\geqslant$ 30 min. Persistently long WASO, as measured by actigraphy, was associated with elevated SBP, after controlling for covariates.
Conclusions: Consistent with anecdotal evidence, it appears as if a subset of women report substantial difficulty initiating and maintaining sleep during early pregnancy and this may augment the risk of higher BP and BMI. Understanding these relationships is important as CM risk factors are linked to maternal and infant morbidity. Assessing sleep in early pregnancy may bestow time necessary for appropriate intervention.

1. Introduction

Cardiometabolic (CM) disturbances during pregnancy, such as hypertension and obesity, have become increasingly prevalent problems in the United States. Hypertension impacts 6–8% of all pregnancies, while obesity rates have reached upwards of 50% in reproductive-aged women [1]. Hypertensive disorders in pregnancy increase the risk of abruptio placentae, disseminated intravascular coagulation, cerebral haemorrhage, hepatic failure and acute renal failure [2], all of which are potentially deadly disorders to both mother and foetus. Elevated blood pressure (BP) in pregnancy can also be part of pre-eclampsia and eclampsia, which carry maternal mortality rates of up to 10–15% [3]. In addition, hypertensive disorders that develop in pregnancy impart a risk of maternal cardiovascular disease later in life [4]. Obesity in pregnancy is also associated with maternal morbidity, including gestational and later life diabetes, hypertensive disorders and higher rate of caesarean delivery [5,6]. Likewise, maternal obesity is associated with foetal morbidity, such as foetal macrosomia and foetal anomalies, including neural tube defects and cardiac malformations [7].

Sleep is a common behavioural pathway associated with both hypertension and obesity. Sleep disturbances, including short/long sleep, poor sleep quality and poor continuity, are associated with hypertension and obesity in non-pregnant cohorts, even after controlling for traditional risk factors such as age, smoking and race [8]. Recent studies have observed similar associations in pregnant women. For example, Williams et al. [9] reported that both long and short sleep duration in early pregnancy were associated with elevated BP in later pregnancy. Likewise, Althuizen et al. [10] identified short sleep as a correlate of excessive weight gain in pregnancy. However, they only assessed sleep in later pregnancy (30 weeks). We speculate that poor sleep in early gestation

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(operationalised as <20 weeks) may contribute to dysregulation in the CM pathways resulting in hypertension and obesity, thereby increasing the risk of adverse pregnancy outcomes [9].

Sleep can be measured subjectively or objectively using various methods. Subjective methods include questionnaires and sleep diaries, whereas objective methods include actigraphy and polysomnography (PSG) [11]. Actigraphy is a convenient and reliable adjunct to questionnaire data. At the present time, few studies have ascertained both subjective and objective sleep in pregnant women. Previous studies using actigraphy in pregnancy have been limited by short collection periods (<72 h) and small cohorts (<50) [12]. Thus, the purpose of this preliminary study was to (1) examine whether women with poor sleep, defined as short sleep duration, longer wake after sleep onset (WASO) or longer sleep onset latency (SOL) at 10–12 weeks, measured by both sleep diary and actigraphy, would have higher BP or higher BMI across early pregnancy (10–20 weeks) and (2) whether women with persistent sleep disturbance (i.e., poor sleep between 10 and 20 weeks) would have greater increases in BP or body mass index (BMI) compared to women with intermittent sleep disturbance or no sleep disturbance.

2. Methods

2.1. Participants

Participants assessed for this secondary analysis were drawn from a prospective, observational study (Sleep in Pregnancy (SLIP)) [13], in which sleep disturbance and inflammation in early gestation are evaluated for their role in adverse pregnancy outcomes. Women in general good health were recruited at approximately 10 weeks of gestation by self-referral, physician referral, local advertising or participation in university research registries. Exclusion criteria included a current diagnosis of depression, bipolar disorder or anxiety, self-reported sleep disorders (e.g., sleep apnoea, narcolepsy, hypersomnia or insomnia), current use of an antidepressant, antipsychotic or anti-inflammatory medication, gynaecologic anatomical abnormality, hypertension, diabetes, HIV or other major chronic diseases. All participants provided written informed consent and the study was approved by the University of Pittsburgh Internal Review Board. Participants received monetary compensation for their participation.

2.2. Study protocol and procedures

At enrolment, participants provided information on current health, health behaviours and socio-demographic information. Sleep data were collected T1 (10–12), T2 (14–16) and T3 (18–20 weeks of gestation). Eligible participants completed the Pittsburgh sleep diary [14] daily and wore a wrist actigraph (Phillips/Respironics, Murrysville, PA, USA) for 14 consecutive days, 24 h a day during each assessment period. On day 15, during each measurement cycle, participants presented for a health assessment, including BP and weight measurements.

2.3. Sleep

Daily sleep diaries [14] were completed by participants for three 2-week periods. Participants recorded information about nocturnal sleep upon awakening and about daytime information prior to retiring for the day. Participants concurrently wore a wrist actigraph (Actiwatch64, Philips Respironics, Bend, OR, USA) on the non-dominant wrist. The actigraph continuously monitors movement and activity in 1-min epochs via an accelerometer and storage chip. Actigraphy data were scored using Actiware-Sleep 5.0 analysis software (Actiwatch64, Philips Respironics, Bend, OR, USA).

Measures of sleep included subjective and actigraphy-assessed indices of (a) SOL, the amount of time between reported bedtime and sleep onset time; (b) WASO, the amount of time awake after sleep onset; and (c) *total nocturnal sleep time* (TST), defined as the amount of time between sleep onset and sleep offset. All measures were averaged across all available nights for each period (up to 14 days). Sleep variables were examined as continuous or categorical as appropriate. The three sleep variables were categorised to depict the presence or absence of sleep disturbance using published data from pregnant women in early pregnancy [9,15]. SOL was considered disturbed if the diary or actigraphy 2-week average was $\geqslant$20 min. WASO was considered disturbed if the diary or actigraphy 2-week average was $\geqslant$30 min. TST was considered disturbed if the diary 2-week average was <7 h or if the actigraphy 2-week average was <6 h. We chose these two different TST cut-offs to maintain similar cell sizes, as approximately equal percentages were represented from each method (24% from diary-assessed <7 h and 20% from actigraphy-assessed <6 h). In addition to the cross-sectional groupings, we further categorised participants into 'persistent' (had sleep disturbance at all time points), 'intermittent' (has sleep disturbance at one or two time points) and 'no' (has no disturbed sleep) sleep disturbance groups.

2.4. Outcomes measures

BP assessments were taken using a WelchAllyn® automated sphygmomanometer while participants were in a seated position. Sitting values of systolic blood pressure (SBP) and diastolic blood pressure (DBP) were collected by the study nurse. *Weight* was assessed using a Detecto® electronic scale and is reported in pounds. *Height* was assessed using a Seca® electronic measuring rod. Both measures were taken with no shoes on. *BMI* was calculated as kg m^{-2}. All measures were collected thrice during the study period: 12, 16 and 20 weeks.

2.5. Covariates

Potential covariates included socio-demographics and indices of health behaviours previously associated with BP or weight gain/BMI during pregnancy. *Age*, *race* (Caucasian or African American/Other), *marital status*, *exercise* (yes or no) and *number of children < 18 years at home* (0 or >1) were established by self-report. *Pre-pregnancy weight* was collected by a nurse at the first clinical visit. Self-reported symptoms of *stress* were measured with the 10-item Perceived Stress Scale (PSS 10) (Cohen et al., 1983), which was calculated as a continuous variable. Self-reported symptoms of depression were measured with the Inventory of Depressive Symptoms (IDS) questionnaire [16]. The IDS, minus sleep items, was calculated as a continuous variable.

2.6. Statistical analyses

Descriptive statistics were examined to characterise the demographics for the total cohort and by diary and actigraphy sleep measures. Means and standard deviations of all continuous measures, by time point, for the total sample are reported. Examination of normal distribution assumption for continuous data was determined by *q–q* plots, histograms and the Shapiro–Wilk test. Counts and percentages for categorical data are reported. Pearson's or Spearman's rank correlation analyses were conducted to assess the degree of linear relationship between continuous variables, as applicable. Partial correlations adjusted by age, race, marital status, number of children, stress and depression were also calculated. Point biserial and polyserial correlation analyses were conducted to assess the degree of relationship between dichotomous and continuous measures, or ordinal and continuous measures,

Table 1
Subject demographics.

	Mean ± SD	N
Age (yrs)	29 ± 5 Range (19–40)	161
Pre-pregnancy weight (lbs)	159.5 ± 40.0 Range (97.0–332)	119
Weight at T1 (lbs)	159.9 ± 40.4 Range (88.4–343.5)	154
BMI at T1 (kg/m^2)	26.6 ± 6.2 Range (15.2–55.2)	151
Blood Pressure at T1		
Diastolic	67.6 ± 8.5 Range (44–92)	151
Systolic	108.0 ± 12.6 Range (80–150)	151
Perceived stress scale	13.9 (6.3) Range (0–33)	
Inventory of depressive symptoms (sleep item removed)	4.9 (3.0) Range (0–19)	
	N (%)	
Race		
Caucasian	113 (70.2)	
African-American/Other	48 (29.8)	
Marital status		
Married	104 (65.8)	
Living with partner	27 (17.1)	
Divorced	1 (0.6)	
Never married	26 (16.5)	
Number of children		
0	96 (61.9)	
1	44 (28.4)	
2	10 (6.5)	
3+	5 (3.2)	

respectively. Sleep measures were dichotomised to define 'good' and 'poor' sleep. Diary measures of poor sleep were defined as follows: SOL ($\geqslant$20 min), WASO ($\geqslant$30 min) and TST (<7 h). Poor sleep for actigraphy measures were defined as: SOL ($\geqslant$20 min), WASO ($\geqslant$30 min) and TST (<6 h). Frequencies of 'good' and 'poor' are reported to further describe the degree of poor sleep of each sleep variable at each time point and across time. Mixed modelling techniques were used to examine whether binary measures of sleep were associated with BMI and BP. The SAS procedure MIXED was used for modelling the main effects of time and sleep, and time by sleep interactions, and to account for within-subject correlation. For these models, logarithm base 10 transformations were used for BMI. These models were also adjusted for potential confounders including age, race, marital status, whether there was a child at home, IDS with sleep item removed, PSS, pre-pregnancy weight and exercise. All analyses were conducted using SAS, version 9.3 statistical software (SAS Institute Inc., Cary, NC, USA).

3. Results

Participant characteristics are presented in Table 1. Participants (n = 161) were 29 ± 5 years of age and 70.2% of participants self-identified as Caucasian. Over 80% were married or living with a partner and over 37% had at least one child <18 years of age living at home. BPs of these women reflected the healthy status. Only two women had high BP at 12 weeks (>140/90). Table 2 presents the sleep parameters at baseline (10–12 weeks), T2 (14–16 weeks) and T3 (18–20 weeks). Evaluation of the covariates indicated that pre-pregnancy weight was correlated with T1 SBP (r (93) = 0.46, p < 0.0001) and DBP (r (93) = 0.39, p = 0.0002) after adjusting for age, race, marital status, number of children, stress and depressive symptoms. Women who were married had shorter diary and actigraphy-assessed SOL (r (159) = −0.28, p = 0.0003; r (146) = −0.24, p = 0.0038) and actigraphy-assessed WASO (r (146) = −0.33, p < 0.0001) and longer actigraphy-assessed sleep duration (r (146) = 0.26, p = 0.0012). Having more children at home was also associated with longer diary-assessed SOL (T1: r (153) = 0.22, p = 0.0138), greater weight (T1: r (148) = 0.23, p = 0.0119) and higher BMI (T1: r (145) = 0.24, p = 0.0102), but not SBP or DBP. Caucasian women had shorter SOL (diary and actigraphy) (T1: r (159) = −0.27, p = 0.0005; r (146) = −0.24, p = 0.0035), less actigraphy-assessed WASO (T1: r (146) = −0.29, p = 0.0004) and longer actigraphy-assessed sleep duration (T1: r (146) = 0.32, p < 0.0001).

The next set of correlations assessed whether sleep at T1 (10–12 weeks) was associated with any of the CM factors at each time point (T1, T2 or T3). Diary-assessed SOL at T1 was associated with SBP at T2 after adjusting for covariates (r (132) = 0.18, p = 0.0329). However, this trend disappeared after correcting for multiple comparisons. Neither diary- nor actigraphy-assessed WASO and TST at T1 were associated with any of the CM factors.

We further evaluated the frequency of disturbed sleep at each time point, and whether sleep disturbance was present, absent or intermittent across all time points. Table 3 presents the frequencies of women who met one criterion at each time point, as well as the number and percentage of women who met criteria for persistent, intermittent or no sleep disturbance for each sleep variable. Considering SOL, for instance, we identified 22 (17%) women who had persistent SOL $\geqslant$ 20 min as measured by diary, yet only six (6%) met similar criteria based on actigraphy. Twenty-five (19%) women had long SOL intermittently as measured by diary, with 12 (11%) meeting similar criteria as measured by actigraphy. In contrast to previous reports [15,17], over half of the women, 71 (54.6%), had SOL < 20 min based on diary, as well as for actigraphy-assessed SOL, 72 (68%), indicating no concerns with sleep initiation. Table 4 presents the frequencies at each time point of women who met two or three criteria.

Finally, we examined whether persistent sleep disturbance (present at all time points) was differentially associated with the CM factors. For diary-assessed SOL and WASO, there was a statistically significant interaction between time and sleep group (SOL: F(2,128) = 3.67, p = 0.028; WASO: F(2,128) = 3.12, p = 0.047) and a significant within-subject main effect of time (SOL: F(2,128) = 164.16, p < 0.0001; WASO: F(2,128) = 180.16, p < 0.0001) for

Table 2
Sleep characteristics at all time points.

Sleep Measure (min)	N	T1 (10–12 weeks) Mean (SD)	N	T2 (14–16 weeks) Mean (SD)	N	T3 (18–20 weeks) Mean (SD)
Diary						
SOL	161	20.0 (14.9)	138	15.7 (12.6)	133	16.8 (12.8)
WASO	161	24.2 (18.2)	138	20.5 (16.6)	133	20.1 (14.3)
TST	161	460.9 (52.0)	138	459.3 (55.7)	133	454.1 (53.8)
Actigraphy						
SOL	148	14.7 (11.4)	124	12.5 (9.0)	122	12.9 (10.3)
WASO	148	90.8 (56.6)	124	78.5 (42.6)	122	71.5 (37.6)
TST	148	390.0 (63.5)	124	390.5 (57.9)	122	395.9 (50.8)

SOL = Sleep onset latency; WASO = Wake after sleep.
Onset; TST = total sleep duration.

Table 3
Frequency of women who met individual criteria for sleep disturbance.

Sleep Measure	T1 *N* (%)	T2 *N* (%)	T3 *N* (%)	Persistent sleep disturbance *N* (%)	Intermittent sleep disturbance N (%)	No sleep disturbance *N* (%)
Diary						
SOL ⩾ 20 min	60 (37.3)	34 (24.6)	39 (29.3)	22 (17.0)	25 (19.2)	71 (54.6)
WASO ⩾ 30 min	44 (27.3)	34 (24.6)	23 (17.3)	14 (11.0)	28 (21.5)	79 (60.8)
TST < 7 h	39 (24.2)	28 (20.3)	35 (26.3)	15 (12.0)	18 (13.9)	83 (63.9)
Actigraphy						
SOL ⩾ 20 min	31 (21.0)	20 (16.1)	22 (18.0)	6 (6.0)	12 (11.3)	72 (67.9)
WASO ⩾ 30 min	145 (98.0)	121 (97.6)	116 (95.0)	96 (91.0)	1 (.94)	0 (0)
TST < 7 h	38 (25.7)	36 (29.0)	25 (20.5)	13 (12.0)	16 (15.1)	67 (63.2)

Persistent sleep disturbance = poor sleep at T1, T2 and T3.
No sleep disturbance = no poor sleep at T1, T2 and T3.
Intermittent sleep disturbance = poor at T1 but not T2 and T3, or poor at T2 but not T1 and T3, or poor at T3 but not T1 and T2.

Table 4
Frequency of women who met two or more criteria for sleep disturbance.

Sleep Measure	T1 *N* (%)	T2 *N* (%)	T3 *N* (%)
Diary			
SOL ⩾ 20 min + TST < 7 h	18 (11.2)	11 (8.0)	16 (12.0)
WASO ⩾ 30 min + TST < 7 h	15 (9.3)	11 (8.0)	12 (9.0)
SOL ⩾ 20 min + WASO ⩾ 30 min	23 (14.3)	14 (10.1)	14 (10.5)
Actigraphy			
SOL ⩾ 20 min + TST < 6 h	15 (10.1)	10 (8.1)	7 (5.7)
WASO ⩾ 30 min + TST < 6 h	38 (25.7)	35 (28.2)	23 (18.9)
SOL ⩾ 20 min + WASO ⩾ 30 min	31 (21.0)	20 (16.1)	22 (18.0)
All Diary-assessed criteria	9 (5.6)	7 (5.1)	8 (6.0)
All Actigraphy-assessed criteria	15 (10.1)	10 (8.1)	7 (5.7)

unadjusted models, revealing that changes in BMI over time are not the same for all groups. These effects remained statistically significant following covariate adjustment for age, race, marital status, exercise, having children at home, the IDS with the sleep item removed, the PSS and pre-pregnancy weight. Only pre-pregnancy weight was a statistically significant correlate of BMI or BP ($P < 0.001$). Figs. 1 and 2 show that BMI increases over time for both groups (persistent SOL/WASO and no persistent SOL/WASO). Interestingly, women with intermittent long WASO showed a greater increase in BMI over time. Lastly, for actigraphy-assessed WASO and SBP, there was a significant within-subject main effect of time ($F(2,112) = 4.79$, $p = 0.0101$) and a significant interaction between the time and sleep groups ($F(2,112) = 3.33$, $p = 0.0394$) after controlling for covariates (Fig. 3). When evaluating sleep measures as continuous data, diary-assessed WASO and TST and actigraphy-assessed SOL, WASO and TST were not associated with BMI.

4. Discussion

This study evaluated sleep collected concurrently via diary and actigraphy in early pregnancy and two particularly relevant CM risk factors, BP and BMI. We found that issues with diary-assessed SOL and WASO (two measures of sleep continuity) were associated with higher BP and/or BMI, which remained significant after controlling for covariates. However, given the cross-sectional nature of the BP and BMI data, we encourage the reader to interpret the findings with caution. Total sleep time (TST) was not associated with either of the CM risk factors. This is in contrast to a report by Williams et al. [9]. However, our BP assessments were in early pregnancy when BP is typically lower than pre-pregnancy values or in later pregnancy [18]. To our surprise, we observed no significant associations among the actigraphy-assessed variables and BP or BMI. While actigraphy has been validated in the general population, it has yet to be thoroughly studied in pregnant women. Actigraphy determines sleep or wake by measuring nocturnal movement. In pregnancy, there are multiple factors that may cause a woman to have more movement during sleep without actually awakening [19]. Further studies are needed in order to better interpret actigraphy data in the pregnant population.

Several studies have noted an association between sleep disturbance, especially poor sleep quality, and higher BP [8,20]. We noted a positive relationship between (T1) actigraphy-assessed WASO ⩾ 30 min and BP across all time points, and this association remained significant after adjusting for covariates. Furthermore, we found that women with longer WASO did not exhibit the typical decrease and gradual rise in BP that is often observed in the later part of the second trimester. BP fluctuates in women entering the second trimester due to a series of normal vascular adaptations [18]. Our findings indicate that women with longer WASO show a blunted change in their BP when compared to those with average WASO (Fig. 3). While suggestive, further studies, including 24-h ambulatory BP measurements, are needed to better understand the patterns and mechanisms associated with sleep disturbance and changes in BP, and how this may impact health in later pregnancy. Moreover, the criteria used to determine irregular BP values may not be sufficient to detect a signal. Future studies are needed to determine optimal cut-off values for defining abnormal sleep as well as critical BP values in early pregnancy.

The relationship between SOL and BMI was diminished to a trend after controlling for the covariates of age, race, marital status, having children at home, stress, depressive symptoms and pre-pregnancy weight. This finding is consistent with the published literature noting that stressful life events are associated with specific aspects of sleep disturbance, like long SOL [21]. Additionally, several studies have shown that long WASO, as well as short sleep duration, are linked to higher BMI [22,23]. Our findings do not support previous findings. However, this may be a reflection of the population under study. Women are expected to gain weight during pregnancy [24] and report of substantial sleep disturbance is considered the norm [25]. The association between SOL and BMI is in line with the hypothesis that sleep disturbance is associated with increased BMI [8,26]. We speculate that women who report disturbances in several domains of sleep, including difficulty initiating and maintaining sleep, as well as shorter sleep duration and poor quality, may be at the greatest risk of gaining excessive weight or developing high BP. The cell sizes were insufficient to adequately address this question. While all women gain weight during pregnancy, it is the degree of change in relation to pre-pregnancy BMI that is particularly relevant to risk of adverse outcomes [27,28]. When analysing the degree of change in BMI across time, women with longer SOL had higher BMI at each time point. Our data also suggest that women who have longer SOL also experienced more stress than their counterparts. Therefore, it is possible that stress via sleep disturbance may account for the increased

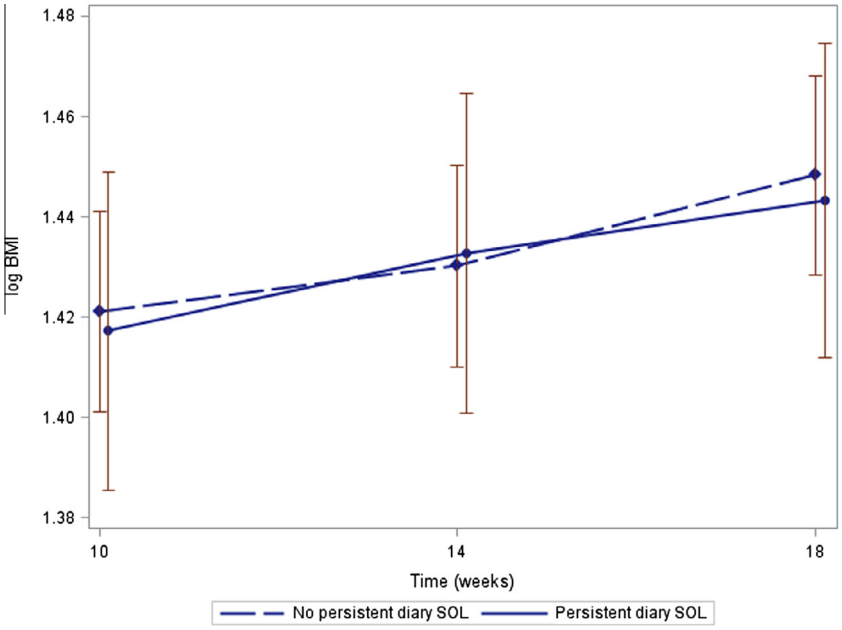

Fig. 1. Estimated logarithm body mass index (log BMI) values for women with persistent long diary-assessed SOL ($\geqslant$20 min, solid line) and without persistent long diary-assessed SOL (<20 min, dash line) are expected means from the linear mixed-effects model with covariate adjustment for age, race, marital status, whether she has a child at home, IDS with sleep item removed, PSS, pre-pregnancy weight and exercise. Error bars indicate 95% confidence intervals. Linear mixed-effect model analysis showed significant main effect for time ($F(2,69) = 127.09$, $p < 0.0001$). Results show that BMI is increasing over time for both sleep groups (persistent SOL and no persistent SOL).

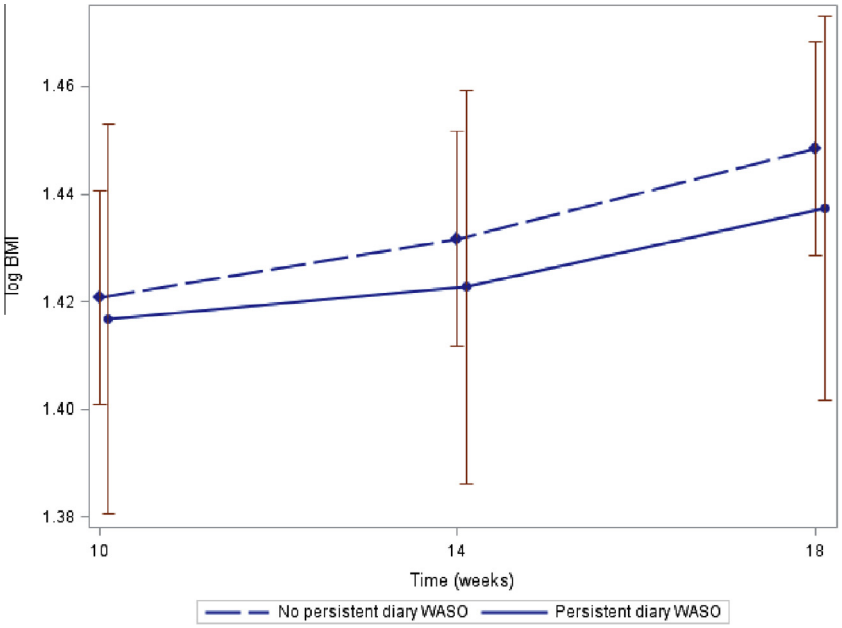

Fig. 2. Estimated logarithm body mass index (log BMI) values for women with persistent long diary-assessed WASO ($\geqslant$30 min, solid line) and without persistent long diary-assessed WASO (<30 min, dash line) are expected means from the linear mixed-effects model with covariate adjustment for age, race, marital status, whether she has a child at home, IDS with sleep item removed, PSS, pre-pregnancy weight and exercise. Error bars indicate 95% confidence intervals. Linear mixed-effect model analysis showed significant main effect for time ($F(2,69) = 138.62$, $p < 0.0001$). Results show that BMI is increasing over time for both sleep groups (persistent WASO and no persistent WASO).

BMI. Not surprisingly, pre-pregnancy weight was a significant correlate in both models examining BMI. The degree to which women gain weight following conception is a strong determinant of future maternal morbidity [28] Women who have low net gestational weight gain are also at a risk of adverse pregnancy outcomes. Further evaluation of these bidirectional relationships is warranted.

Lastly, given the evidence that chronic sleep disturbance, rather than cross-sectionally measured sleep, is associated with greater morbidity [29], we identified those women who had persistent sleep disturbance and compared them to women with no sleep disturbance. Although the cell sizes were not powered to detect a difference, we did observe that women with persistent SOL $\geqslant$ 20 min had higher BMI values at all time points. Additional studies that follow women throughout pregnancy are needed to determine whether chronic sleep disturbance is necessary to substantially increase the risk of morbidity.

These findings add to the growing evidence that sleep disturbance in pregnancy may play a role in adverse pregnancy outcomes [30]. In accordance with previously published studies, our data suggest that disturbed sleep as early as 10–12 weeks of gestation may affect CM risk factors known to adversely affect health and outcomes [9]. We do however acknowledge some limitations

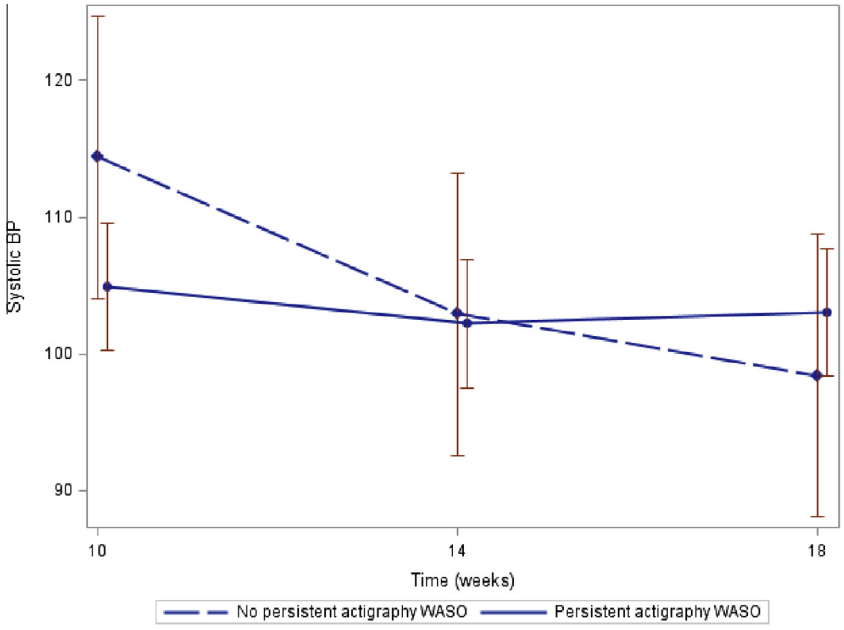

Fig. 3. Estimated systolic blood pressure (Systolic BP) values for women with persistent long actigraphy-assessed WASO (⩾30 min, solid line) and without persistent long actigraphy-assessed WASO (<30 min, dashed line) are expected means from the linear mixed-effect model with covariate adjustment for age, race, marital status, whether she has a child at home, IDS with sleep item removed, PSS, pre-pregnancy weight and exercise. Error bars indicate 95% confidence intervals. Linear mixed-effect model analysis showed significant main effect for time ($F(2,112) = 4.79$, $p < 0.0101$) and significant interaction between sleep group and time ($F(2,112) = 3.33$, $p = 0.0394$). Results show that systolic BP decreases over time for the no persistent actigraphy-assessed WASO group, and remains constant across time for the persistent actigraphy-assessed WASO group.

in the sample population and measures which temper our interpretations. While some may argue that PSG is the gold standard with which to record sleep information, we contend that the use of actigraphy in this study is a better measurement of sleep. Unlike PSG, actigraphy affords the opportunity to capture change over time and evaluate variability in pregnant sleep. Another limitation is that we only assessed BP once per visit rather than the recommended two or more readings separated by 2 min [31]. We also did not have data after 20 weeks. It is feasible that we would see other associations if we had data from across the entire period of pregnancy. Moreover, a single BP measurement does not allow for the examination of diurnal variation. However, few studies have collected 24-h ambulatory BP in pregnant women due to high participant burden [32]. Future studies need to utilise this method to account for nocturnal BP and associated dipping. Nevertheless, all physiologic measurements were taken between 7:00 am and 10:00 am to control for time effects. Finally, we sought to recruit healthy, low-risk women in order to mitigate the effects of various co-morbidities. However, we were limited to self-report of sleep disorders, psychopathology, other illnesses and medication use. Reliance on self-report can result in some participants entering the study with an undiagnosed condition, which could influence the findings. Particularly relevant here is the absence of data regarding snoring or sleep-disordered breathing (SDB) at each of the time points. While only approximately 10% of pregnant women have SDB in early pregnancy [33], estimates of frequent snoring are around 20% [30], Hence, future studies need to assess snoring and/or SDB in early pregnancy studies on sleep.

This study lays the groundwork for future endeavours assessing sleep in pregnancy and sheds light on the importance of clinical evaluation of sleep patterns in early pregnancy. The significant morbidity associated with elevated maternal BP and BMI make early detection important to avoid subsequent morbidity. One pertinent aspect of sleep is that it is a modifiable behaviour. A growing literature confirms the utility of behavioural interventions to ameliorate sleep disturbances [34]. While this has not been evaluated in pregnant women, we speculate that this option may be especially pertinent given that there are few interventions that can be implemented in pregnancy. A better understanding of the role sleep may play in the development and/or progression of maternal hypertension and unhealthy BMI during pregnancy may prove beneficial for the incorporation of future screening, evaluation and treatment of these conditions.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2014.01.003.

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